CASE REPORTS

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Traumatic Hemoglobinuria Associated with Conga Drumming

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SINCE THE FIRST DESCRIPTION by Fleischer in 1881[1] several cases of march hemoglobinuria have been reported. A causal relationship between running on hard surfaces and hemoglobinuria was suggested by Attlee,[2] but it was Davidson[3] who showed that hemolysis was caused by mechanical trauma to circulating erythrocytes in the plantar blood vessels. This simple explanation has been confirmed by Buckle[4] and Spicer;[5] no cellular defect has been isolated. Traumatic hemoglobinuria involving the upper extremities has been encountered less commonly than march hemoglobinuria. In 1903 Ensor and Barratt[6] reported hemoglobinuria in a demented patient who was accustomed to violent bouts of head slapping. More recently, hemoglobinuria has been shown to occur after karate exercises[7] and after conga drum playing.[8]

We report here three persons in whom traumatic hemoglobinuria occurred after conga drumming. Schistocytosis was also present in one, and glucose-6-phosphate dehydrogenase (G-6-PD) deficiency in another.

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Reports of Cases

CASE 1. A 31-year-old black man, a college student, was seen at the University of California Ambulatory and Community Medicine Clinic because of episodic passing of dark urine. Four weeks earlier he had enrolled in a conga drumming class, and since then he had noted cola-colored urine after each strenuous, bare-handed drumming session. The urine returned to normal color within a few hours. He reported using only an oral decongestant compound (Allerest®) occasionally and marijuana infrequently.

On physical examination, the patient was found to be healthy-appearing and muscular, with normal vital signs. The palmar surfaces of the hands were normal, there was no hepatosplenomegaly and posture was normal. Findings in the remainder of the examination were unremarkable.

A sample of urine that the patient brought with him was qualitatively strongly positive for protein and hemoglobin (Labstix) and appeared black. Findings on analysis of a fresh urine specimen, hematocrit determination and a blood smear were within normal limits, but the reticulocyte count was 3.5 percent. A leukocyte count and differential count, erythrocyte sedimentation rate (Westergren), serological test for syphilis (Venereal Disease Research Laboratories—VDRL—reaction), liver function tests and determinations of blood urea nitrogen, serum protein and albumin, and total and direct bilirubin gave findings within normal limits. Serum creatinine was 1.3 mg per 100 ml (normal up to 1.2 mg per 100 ml), creatinine clearance was normal at 144 ml per minute and plasma hemoglobin was less than 5 mg per 100 ml (normal). Results of hemoglobin electrophoresis, direct and indirect Coombs' tests, Hams' test, glucose-water test and porphyrin screen of urine were negative or normal. Glucose-6-phosphate dehydrogenase was 1 international unit (IU) per gram of hemoglobin (normal 4 to 8 IU). Findings on a chest x-ray study and an intravenous pyelogram were unremarkable. There was no growth on urine culture. On examination of stool, Giardia lamblia cysts were noted.

The giardiasis was treated with metronidazole (Flagyl), 200 mg three times daily for one week.

The patient was informed of the probable reason for the hemoglobinuria and was advised to maintain a high intake of fluids at each drumming session.

CASE 2. A healthy 32-year-old black man, a medical student, had recently noted dark brown urine after conga drumming sessions. He was a conga drummer of 17 years' experience and had played professionally. Approximately six months previously he noted dark brown urine 30 minutes after a drumming session and again two hours later. Though he sensed a fullness in both flanks for two days after the session, findings on urinalysis on the first day were reported to be normal by a college physician. Since then the patient has noticed dark urine three times, always after strenuous drumming sessions. In the recent past he had participated actively in swimming and karate (without board-breaking exercises), but no dark urine had been noted. He used no medications or street drugs.

On physical examination, the patient's vital signs were normal. Flat feet, tinea pedis and an associated palmar id reaction were noted. The palmar surfaces were otherwise normal. There was no enlargement of abdominal organs, and posture was normal. The remainder of the physical examination was not remarkable. Findings on analysis of a fresh urine specimen, hematocrit determination, blood smear, leukocyte count and differential count were within normal limits. Reticulocyte count was 1.9 percent. A VDRL reaction test and determinations of blood urea nitrogen, serum creatinine, serum protein and albumin, and total and direct bilirubin gave either negative or normal findings, as did hemoglobin electrophoresis, a direct and indirect Coombs' test, a Hams' test, a glucose-water test and a glucose-6-phosphate dehydrogenase determination. No abnormalities were noted on an x-ray film of the chest.

CASE 3. A healthy 29-year-old black man, an acquaintance of the patient in Case 2, had occasionally noticed dark urine after particularly strenuous drumming sessions. He was an amateur conga drummer of 17 years' experience. He could not recall when he first passed dark urine in association with drumming, but was sure that it was always after his "heaviest jam sessions." Because he had recently experienced such an episode, he agreed to take part in the drumming session (described below) but declined any blood tests other than those described. He occasionally used marijuana, lysergic acid diethylamide and amphetamine sulfate.

On examination, vital signs and posture were found to be normal. There was no enlargement of abdominal organs. The palmar surfaces were normal, and the remainder of the physical examination was unremarkable. The hematocrit determination, blood smear and reticulocyte count gave findings within normal limits. Results of a leukocyte count and differential count, a sickle-cell screen and a glucose-6-phosphate dehydrogenase determination were negative or normal.

Materials and Methods

Drumming Session

A drumming session was arranged for the three subjects in order to carefully investigate the hemoglobinuria. At the time of this session it had been six months, one month and two weeks respectively since Patients 1, 2 and 3 had first been seen. Hemoglobinuria had abated in Patient 1 during this time and now occurred only after long, strenuous sessions. All three had refrained from drumming for five to seven days and none had had any medications for a week before the session.

Blood was withdrawn from an antecubital vein and transferred to plain and ethylenediamine tetraacetic acid (EDTA) treated containers, and urine specimens were collected before and after the drumming session. Patients 1 and 3 played in their accustomed sitting positions, whereas Patient 2 stood. All drums were of standard conga styling; the playing surface of each was thick animal skin cured to wood-hard consistency. The drums rested on a carpeted floor and were held in place by metal stands. Each patient had fluids as he desired during the drumming session. The session lasted for 2½ hours and consisted of 20-minute drumming periods alternated with 10-minute rests.

Laboratory Methods

Serum hemoglobin levels were measured by the benzidine-peroxidase method,[9] serum haptoglobins by the Beckman microzone technique,[10] and serum creatine phosphokinase and lactic dehydrogenase by the kinetic rate method.[11,12]

After concentration by ultrafiltration in collodion bags, urine specimens were qualitatively studied by starch gel electrophoresis.[9] Rough quantitation of urine hemoglobin was provided by use of a commercially produced test (Hemastix). Protein was quantitated by the sulfosalicylic

acid method and was graded from 0 to ++++. A Prussian blue stain was used to test for the presence of hemosiderin. Phenotyping of serum haptoglobin before and after the session was done by starch gel electrophoresis in Tris/citrate-borate discontinuous buffer at pH 8.65.[9] Hematologic review was made of peripheral blood smears prepared with Wright's stain.

Results

In Patient 1, baseline values were normal except for slightly depressed serum haptoglobin (42 mg per 100 ml) (Table 1). Baseline serum haptoglobin in Patient 2 was elevated (181 mg per 100 ml) as was creatine phosphokinase (74 IU). In Patient 3, laboratory values were normal. Patients 2 and 3 had haptoglobin of type 2-1; Patient 1's phenotype was not determined.

The drumming session was without incident, and the three subjects felt well but tired afterwards. Patients 1 and 2 noticed a tingling sensation in their hands; both had associated this in the past with the voiding of dark urine. Urine samples collected immediately after the drumming session showed the expected changes of mild hemoglobinuria (Table 1).

The urine specimens from Patients 2 and 3 contained a wide spectrum of proteins. The heme protein in each had a greater anodal migration than normal hemoglobin A, and it was believed to represent hemoglobin Koellicker.[13] A fainter band with the migration pattern of hemoglobin A was also present in each. Myoglobin was not present in either sample. Urine concentration was not done on the specimen from Patient 1, but hemoglobin was determined by positive benzidine staining after differential precipitation with ammonium sulfate.[9] After drumming, serum hemoglobin increased by 29, 136 and 124 mg per 100 ml, and serum haptoglobin decreased by 91, 90 and 24 mg per 100 ml in Patients 1, 2 and 3, respectively. Serum creatine phosphokinase rose in Patients 1 and 3 and remained slightly elevated in Patient 2. Serum lactic dehydrogenase became abnormal only in Patient 2.

Discussion

As in other patients with traumatic hemoglobinuria, elevated serum hemoglobin and depressed serum haptoglobin were present in these patients after traumatic exercise. In two of them there was grossly pigmented urine and in all there was labo-

TABLE 1.—*Alterations in Plasma and Urine after Conga Drumming for 2½ Hours*

Substance	*Normal Value*	*Patient 1*		*Patient 2*		*Patient 3*	
		Before	*After*	*Before*	*After*	*Before*	*After*
Plasma							
Hemoglobin (mg/100 ml)	0.5-25	7.08	36.6	13.5	149.9	9.9	134
Haptoglobin (mg/100 ml)	50-150	42	33	181	91	103	79
Creatine phosphokinase (IU/liter)	10-62	60	88	74	70	60	140
Lactic dehydrogenase (IU/liter)	20-95	82	75	70	135	90	65
Urine							
Color		Clear yellow	Clear yellow	Clear yellow	Cloudy brown	Clear yellow	Pink
Specific gravity		1.012	1.008	1.018	1.023	1.007	1.004
Albumin*	None	None	Trace	None	+++	None	++
Hemoglobin*	None	None	++	None	++++	None	++++
Leukocytes (per high power field)	None	0-2	1-3	2-10	5-10	0-2	0-3
Erythrocytes (per high power field)	None	None	0-1	None	None	None	0-2
Casts (per high power field)	None	None	0-2 granular	None	Many granular	None	Many granular
Hemosiderin	None	None	None	None	None	None	None

*Graded from 0 to ++++.

ratory evidence of hemoglobinuria. A rise in serum creatine phosphokinase suggested there might be a component of muscle injury in at least two of the subjects, because creatine phosphokinase levels are said not to be affected by hemolysis,[9] and were not elevated in a patient with traumatic hemoglobinuria caused by running.[5] In none of our patients was there demonstrable myoglobinuria. The finding of granular casts is consistent with previous reports of this condition.

It is postulated[4,5,14] that the trauma described in these patients, when added to the pummeling and deformation to which a red cell is normally subjected,[15] is sufficient to cause damage to or total destruction of a small quantity of erythrocytes. It has been estimated that this hemolysis represents only 6 to 40 ml of whole blood.[5,16,17] The resultant free hemoglobin is dissociated into alpha and beta dimers and is bound to the $alpha_2$ globulin, haptoglobin, thus preventing the urinary loss of hemoglobin. If the haptoglobin reserve is overwhelmed by the total serum hemoglobin or, perhaps, by the rapidity of the rise in serum hemoglobin, hemoglobinuria results. Hemosiderinuria may also occur,[18] as may methemalbuminuria.[19,20] We did not find either in our patients.

Although poikilocytosis and "four-leaf-clover" cells have been described in a patient,[21] schistocytosis is not a usual part of traumatic hemoglobinuria. Trace schistocytosis was seen in Patient 2; helmet cells were occasionally seen. The role of glucose-6-phosphate dehydrogenase deficiency, if any, in this disorder is unclear. This deficiency is known to increase susceptibility to hemolysis when affected persons are treated with certain drugs or during intercurrent illnesses.[22] It is not known if there is an increased tendency toward traumatic hemolysis under the conditions described here.

Perhaps the frequency and the potential seriousness of traumatic hemoglobinuria ought to be reappraised. Two-thirds of 26 runners in the 1941 Boston Marathon showed an increase in plasma hemoglobin, and four had hemoglobinuria.[23] In Kaden's report of hemoglobinuria in a conga drummer he stated that other members of the patient's drumming group had noted similar episodes in association with prolonged and violent playing.[8] Our Patient 2 reported he knew of six other cases of pigmenturia associated with conga drumming among his friends. Because of the renewed interest in hand drumming, this problem may be encountered more often in the future. A recent report by Pollard and Weiss,[24] which describes a patient who developed acute tubular necrosis with traumatic hemoglobinuria after a marathon race, emphasizes the potential seriousness of this condition. For this reason there should be an increased awareness of this phenomenon.

Summary

Traumatic hemoglobinuria associated with conga drumming in three young black men was documented during a monitored 2½-hour drumming session. Hemoglobinuria, a mean 10-fold rise in serum hemoglobin values and a mean 38 percent fall in serum haptoglobin were seen after this drumming session. Unexpected and new findings included a glucose-6-phosphate dehydrogenase (G-6-PD) deficiency in one subject and trace schistocytosis in another.

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